

migration speed and cytoskeleton organization. Furthermore, cellular migration is monitored on polymer-tethered bilayer substrates with a sharp boundary or lateral gradient in lipopolymer concentration.

#### 874-Pos Board B629

##### Modeling Follicle Cell Length Oscillations During Tissue Elongation in *Drosophila* Egg Chamber

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Periodic processes are an indispensable part of biological phenomena. Circadian rhythms, heart rhythms, neuronal oscillations, cell cycle, and cytoskeletal structures such as the axonemes of cilia are all examples of systems exhibiting oscillatory dynamics. The underlying mechanisms of several such processes can be explained by understanding the origins of these oscillations and characterizing them. One particular example is the follicle cell basal surface area oscillations observed in *Drosophila* egg chamber during oogenesis. It has been suggested that these oscillations restrict the egg chamber width, and thus help in elongation of the tissue. In this work, we attempt to model these oscillations in follicle cell length using a mechano-chemical model. Our model predicts an increase in oscillation period, upon removal of the basement membrane, which has been observed experimentally upon collagenase treatment of the egg chamber. The model also predicts an inverse relationship of maximum contractile force and oscillation period.

#### 875-Pos Board B630

##### Coupling up: How Interactions between Cell Stresses and Intracellular Biochemistry Affect Cell Spreading

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We present a two-dimensional mathematical model and finite element simulations that allow us to better understand how local cellular deformations must be coupled to the evolution of an intracellular plaque protein that controls the formation of focal adhesions. Specifically, we explore effects of alternate formulations for coupling cellular response to substrate mechanics. Further, we also investigate the effect of initial cellular shape on cell spreading and intracellular stresses. Our aim is to determine whether the initial anisotropy of a cell predisposes it to remain anisotropic during spreading. In addition, we examine the role of focal adhesion strength in maintaining anisotropy. In the models for cell and substrate mechanics we assume that the cell is an active hypoelastic material and the substrate is linearly elastic. Focal adhesions are modeled as a collection of discrete springs that can be added and removed dynamically. This work aims to unearth some of the fundamental mechanisms in cell-substrate interactions.

#### 876-Pos Board B631

##### Calculating Intercellular Stress in a Model of Collectively Moving Cells

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Cells move together in groups during development, wound healing, and cancer metastasis. It remains unclear how collectively moving cells coordinate their motion. In addition to external chemoattractants and exchanging signaling molecules, cells may also respond to mechanical cues. We developed a model of collective cell migration under the assumption that cells align their motility force with the direction of their velocity. This simple mechanism leads to large scale velocity correlations, swirling motion in the bulk of monolayers, and finger-like protrusions at the edge [1]. In experimental studies, the inter- and intracellular stress in the monolayer has been calculated from measured traction forces between the cells and the substrate. Stress builds up successively towards the center of the tissue as the majority of the cells pull outwards [2]. While one dimensional stress profiles are based on a simple force balance, two dimensional stress maps require the additional assumption of an elastic tissue [3], and the validity of this assumption remains disputable. In our model simulations, both the forces on the substrate and the intercellular forces are accessible. We can therefore apply a second method to calculate the stress based on forces between cells. Stress patterns calculated with both methods agree, showing that recovery of the intercellular stress is indeed mostly independent of specific material properties.

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#### 877-Pos Board B632

##### Combination of Chemotaxis and Differential Adhesion Leads to Robust Cell Sorting During Tissue Patterning

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Robust tissue patterning is crucial to many processes during development. The "French Flag" model of patterning by instructive morphogen concentrations has been the most widely proposed model for tissue patterning. However, recently, cell sorting has been found to be an alternative model. In this article, we used computational modeling to show that two mechanisms, namely chemotaxis and differential adhesion, are needed for robust cell sorting. We assessed the performance of each of the two mechanisms by quantifying the fraction of correct sorting, the fraction of stable clusters after correct sorting, time taken for correct sorting and the size variations of the cells having different fates. We found that chemotaxis and differential adhesion confer different advantages to the sorting process. Chemotaxis leads to high fraction of correct sorting whereas differential adhesion leads to high fraction of stable clusters. A combination of both chemotaxis and differential adhesion yields cell sorting that is both accurate and robust. Thus, we propose that both mechanisms are used for cell sorting during tissue patterning in development.

#### 878-Pos Board B633

##### Catching up on Slip: Focal Adhesion Composition and Mechanosensing

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Unlike slip bonds, catch bonds experience reinforcement under tension. Cell adheres to the surface, using integrins forming both catch- and slip- bonds with the surface receptors. How will the catch and slip bonds interact with each other on a single adhesion scale? How does the intracellular structure vary depending on the extracellular matrix stiffness? I discuss the implications of single catch-bond characteristics for the behavior of a load-sharing cluster of such bonds: these are shown to possess a regime of strengthening with increasing applied force, similar to the manner in which focal adhesions become selectively reinforced. In addition, I present numerical simulations of mixtures of catch and slip bonds within single focal adhesion, and propose a model of how they can influence cytoskeletal reorganization, force generation and adhesion growth, interacting indirectly through applied force. Our results may shed new light on the fundamental processes that allow cells to sense the mechanical properties of their environment and in particular show how single focal adhesions may act, autonomously, as local rigidity sensors.

#### 879-Pos Board B634

##### Influence of Substrate Stiffness and Thickness on Cell Traction Forces

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It is known that various cell types can sense and respond to the mechanical properties of their microenvironment. Specifically, cells have been known to spread more when cultured on stiff substrates [1-3] and are able to match their internal stiffness to that of the substrate [2, 3]. Recent works have reported on dynamics of cellular properties such as cell shape, cell spread area, and focal adhesion area, as functions of environmental properties such as substrate stiffness, thickness, and chemistry. Building on earlier models [4, 5, 6], we present mathematical models that enable us to replicate some aspects of experimentally reported time-dependent cell behavior. Our models investigate the adaptation of internal cell stiffness through increase in number of focal adhesion complexes and temporal build-up of traction force. Our models crucially invoke the ability of some cell types to adapt their internal stiffness and show that substrate stiffness and thickness can strongly assist in rapid build-up of traction forces and formation of multiple cooperative focal adhesion complexes. Further using our models we generate some mechanistic insights into why certain cell types under the influence of specific substrate properties exhibit the kind of dynamics that has been